deeper dissection had been carried out, the lump once more mysteriously disappeared without trace. Nevertheless in view of the previous experience it was decided to proceed with the original plan of removal of the lower half of the parotid. The trunk of the facial nerve was identified sufficiently in front of the stylo-mastoid foramen to preserve its blood supply (Patey and Moffat, 1961), and it was then noted that the nerve was unduly superficial and pursued a longer course than normal before dividing into its two main branches. The superfacial parotid—that is, that part of the parotid superficial to the facial nerve—was normal except for fibrosis of the lower pole, presumably the result of the previous biopsy. The lower and posterior half of the superfacial parotid was turned downwards from off the facial nerve and its branches. On pulling on the lower attachment of the superfacial to the subfacial parotid a tumour of the latter with the typical macroscopical appearances of a mixed tumour reappeared rather in the manner of extracting a cork. After mobilizing the facial nerve, the subfacial parotid, including the tumour, was removed in continuity with the normal lower half of the superfacial parotid. On recovery from the anaesthetic the patient showed no facial paralysis, and his post-operative course was uneventful. The pathological examination confirmed the diagnosis of mixed tumour.

DISCUSSION

In light of the operative findings the explanation of the unusual features of this case becomes obvious. This was an example of a mixed tumour developing in the deepest portion of the parotid, a tumour which ordinarily becomes imprisoned in the lateral pharyngeal space to give rise to a bulging of the palate and constitutes the dumb-bell tumour of Patey and Thackray (1957). Presumably in this case the tumour stretched the margins of the space to escape and present superficially. Under the influence of the muscular relaxation of general anaesthesia the tumour twice retreated to its site of origin and apparently disappeared. The case also emphasizes once again that an expanding tumour of the parotid, depending on its site of origin, may push the facial nerve in any direction, and that a tumour which clinically appears superficial may be deep to the facial nerve. The nerve as a result is much nearer to the surface than normal and is thus, unless the possibility is always borne in mind, more vulnerable than normal.

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"Food logistics was critical to the Greek armies during the Peloponnesian war (431 to 404 B.C.). The Athenians lost many decisive battles to the Spartans because of food habits. The battles during this war were conducted mainly on the sea. At mealtimes, the vessels would be beached and the soldiers would journey to the nearest town to buy food for a leisurely repast. On many occasions the Spartans took advantage of this routine. In one instance, the Spartans arranged to have meals for their soldiers at the beach ready for consumption. When the opposing fleets withdrew for mealtime, the Spartans ate quickly and returned to battle. The surprised, confused, and hungry Athenians hastily put out to meet their adversary and suffered major defeat. Much later and less than 200 years ago, the canning process was born out of the Napoleonic Wars when France offered a prize for a method of food preservation which would allow her navy to stay at sea for months and still be well fed. . . . The foods for to-morrow's combat soldier, sailor, and airman will look better, taste better, smell better, and be better preserved." (*New Report*, National Academy of Sciences, National Research Council, September–October, 1962.)

Fatal Marrow Aplasia Complicating Infectious Hepatitis

Major haematological complications in infectious hepatitis are rare, although leucopenia and thrombocytopenia are often seen. Aplasia of the bone-marrow as a result of virus infections is also rarely reported, and only two examples of this condition occurring in infectious hepatitis can be traced in the literature. In the following case fatal marrow aplasia occurred during the course of a typical attack of virus hepatitis.

CASE REPORT

A woman of 64 was admitted to hospital on August 11, 1960. For 10 days she had noticed anorexia, nausea, and the passage of dark urine. One week before admission the stools became pale, and on August 8 she noticed yellowness of the skin. Three days before admission a tendency to bruise was noticed. There was no history of transfusions, of injections, or of contact with any known hepatotoxic agent. She gave a history of angioneurotic oedema, exacerbated by aspirin and treated continuously since 1956 with an antihistamine, meclozine hydrochloride.

Examination on admission revealed icterus, bruising over the sacrum, and petechial haemorrhages over the knees. The liver was palpable half an inch (1.5 cm.) below the costal margin, but the spleen was not felt. The capillary fragility test (Hess) was negative. The haemoglobin was 13.3 g./100 ml. and leucocytes 5,000/c.mm., with a normal differential count. Platelets were 40,000/c.mm. and the bleeding-time (Duke) was 15 minutes. The one-stage prothrombin time (Quick) was normal. The direct Coombs (antiglobulin) test was negative and no platelet antibodies were detected by the mixed antiglobulin technique (Chalmers *et al.*, 1959). The Paul-Bunnell test was negative and no atypical mononuclear cells were seen. The serum total bilirubin was 5.2 mg./100 ml. and the direct van den Bergh reaction was positive. Alkaline phosphatase was 25 King-Armstrong units, thymol turbidity 4.5 units, zinc sulphate turbidity 4.5 units, and serum glutamic oxaloacetic transaminase 180 units. The serum total protein was 8.4 g./100 ml. and electrophoresis showed a reduction in albumin and increases in beta- and gamma-globulin. The urine showed a trace of protein and some bilirubin but no urobilinogen.

Further changes in the serum bilirubin and the haematological findings are shown in the Chart. The sternal marrow on August 18 showed minor abnormalities in the red-cell precursors, many of which contained Howell-Jolly bodies. The myeloid precursors were normal, but there was a decrease in megakaryocytes. A further examination on September 7 showed moderate numbers of red-cell precursors, but myeloid precursors were extremely scanty and no megakaryocytes were seen.

The patient was treated with a 40-g. protein diet and prednisolone was administered from the fifth day in a dosage of 40 mg. daily. The serum bilirubin fell rapidly after the administration of steroids but the petechiae and bruising extended and haematuria developed. A total of 6 pints (3.4 litres) of platelet blood, collected into plastic containers containing sequestrene, was given, with transient and decreasing effect. On September 26 the patient suddenly collapsed and died.

Necropsy Findings.—Apart from multiple petechial haemorrhages and ecchymoses the main finding was massive gastro-intestinal haemorrhage originating in the oesophagus or stomach. The cut surface of the liver showed slight fatty change but little disorganization of structure. Microscopically the liver showed well-preserved architecture but some infiltration of the portal tracts with chronic inflammatory cells. Appearances were compatible with the healing stage of a mild virus hepatitis. The marrow showed groups of erythroid precursors but almost complete absence of myeloid precursors or megakaryocytes.

COMMENT

In some of the earlier reports of thrombocytopenic purpura in hepatitis (Alt and Swank, 1936; Woodward, 1943; Jones and Evans, 1951) the exact nature of the liver disease and the condition of the bone-marrow were uncertain. The case reported by Huguenin *et al.* (1956) developed thrombocytopenia during the course of a mild attack of hepatitis. Liver biopsy showed typical histological changes, the marrow was hyperplastic, and the platelet count rose rapidly to normal. This rapid recovery suggests that cases of this type probably represent merely an exaggeration of the symptomless thrombocytopenia found in many cases of hepatitis by Whitesell and Snell (1949).

Lorenz and Quaiser (1955) described the case of a 9-year-old boy who had a severe hepatitis, the serum bilirubin rising to 32 mg./100 ml. Pancytopenia appeared suddenly seven weeks from the onset, at a time when the hepatic condition was improving. Death from cerebral haemorrhage followed and the bone-marrow at necropsy was aplastic. Kosan (1956) described the case of a man of 22 in whom a severe hepatitis terminated in liver coma after five weeks. Pancytopenia first appeared at the third week and the marrow at necropsy was aplastic. Beickert and Siering (1958) reported a case of moderately severe hepatitis in a 19-year-old man in which pancytopenia appeared during the fifth week. Autoantibodies against red cells, leucocytes, and platelets were detected but there was no response to steroids. Death occurred from cerebral haemorrhage, and at necropsy the marrow was reported to be normal.

The case described here differs from those previously reported since the hepatitis was mild while the marrow damage was severe. The possibility of marrow damage due to drugs was considered, but the only drug administered (meclozine) had been taken continuously, in moderate dosage, for four years and there have been no reports of bone-marrow or liver damage resulting from its use.

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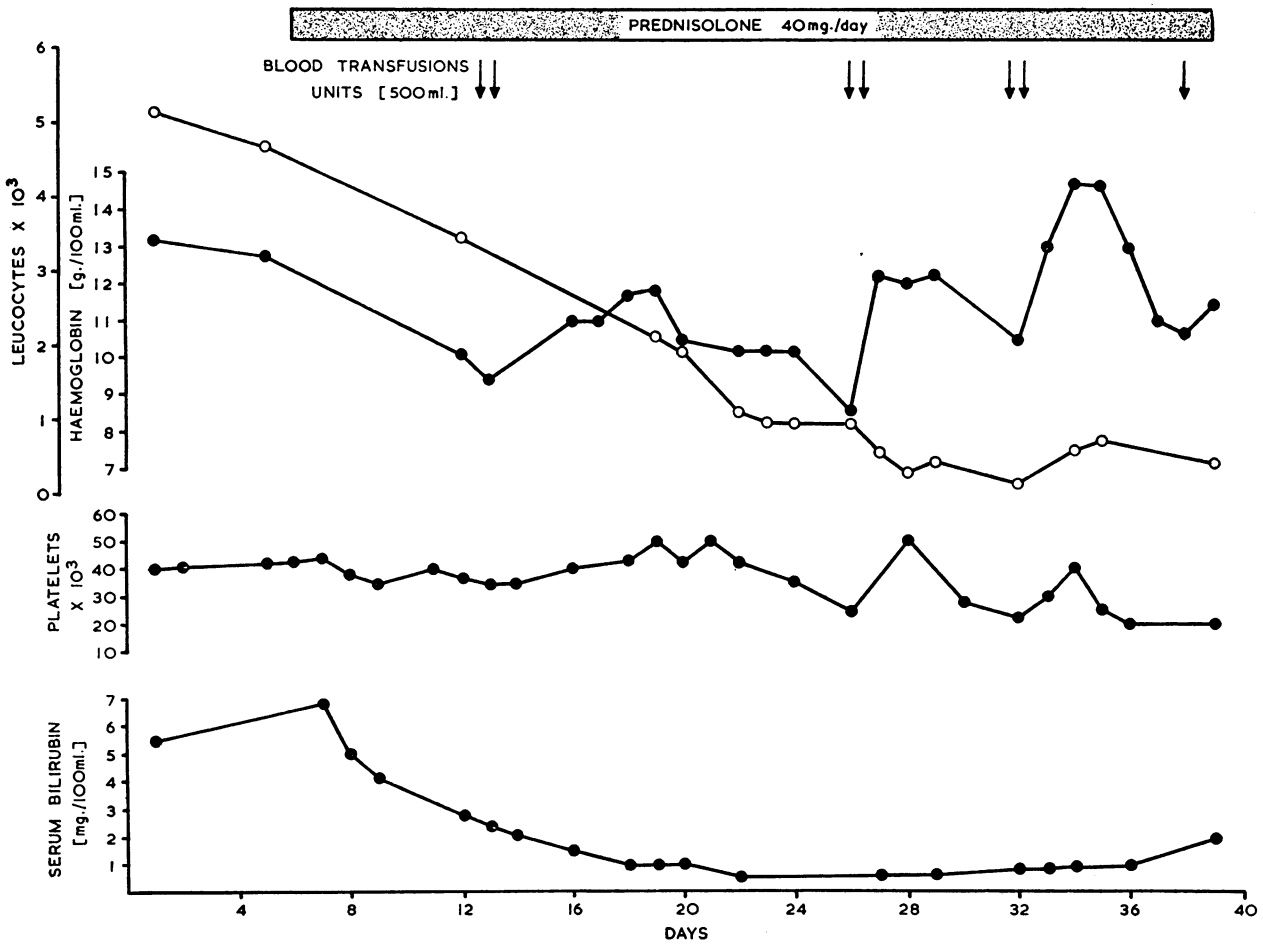

Serial changes in the haematological findings and the serum bilirubin level.